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Local Determinants of Sweating and the Assessment of the "Set Point"

by
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INTRODUCTION

Our laboratory, for a number of years, has been investigating by resistance hygrometry the sudomotor activity of human eccrine sweat glands (Banerjee et al., 1969; Bullard et al., 1967; Bullard et al., 1970). These studies have provided conclusive evidence that the local conditions or the conditions of the microenvironment at the level of the glands can exert considerable influence upon the quantitative responses of the sweat glands to a constant controlled output from the central nervous system. We have presented evidence, for example, that the magnitude of eccrine cyclic sweating in response to a constant central stimulus is significantly altered by the determinants of local skin temperature, local blood flow, local skin hydration, and the degree of acclimation.

Because determinations of hypothalamic, central, or skin temperature thresholds, or set points, for the onset of eccrine sweating during transient experimental conditions of increasing body temperatures tend to give variable results, we have recently utilized the resistance hygrometry technique to study the effects of some of these local determinants upon the assessment of central and skin temperature thresholds for the onset of sudomotor activity.

METHODS

The experiments were performed on 12 healthy male subjects ranging in age from 18 to 33 years and all were experienced in heat exposure studies. The experiments were carried out at different ambient temperatures and cutaneous water loss was continuously measured from small skin areas of 8 cm^2 each by the resistance hygrometry method (Bullard, 1962). In this method dry air was passed over the selected skin areas and the effluent air was passed over resistance hygrometers connected to a Honeywell 1108 Visicorder recording system. A quantitative and continuous record of the onset and time course of local eccrine sweating was obtained under a number of different environmental and local skin conditions.

Rectal temperature was continuously determined by means of a YSI, 401 probe and for the measurement of the tympanic membrane temperature a thin flexible probe with a small (VECO 32A8) thermistor bead was used. Temperatures of different uncovered skin areas were recorded every 32 sec on a 16-point Brown recorder with 24-gauge copper constantan thermocouples.

Several different experimental approaches were used in the study. Different experimental conditions were used to study the effects of local skin temperature,

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the availability of neuroglandular transmitter substance, and the effects of short term acclimation on the latency and corresponding threshold temperatures for the onset of a sweating response to a constant stimulus.

RESULTS

LOCAL SKIN TEMPERATURE AND THE ONSET OF ECCRINE SWEATING. Figure 1 illustrates the effects of local skin temperature upon the onset of eccrine sweating in a resting subject. In these experiments the subjects were initially allowed to equilibrate for 20 min in a cool room maintained at 26°C. As illustrated on the first part of the record no cyclic sweating was recorded from the calf or

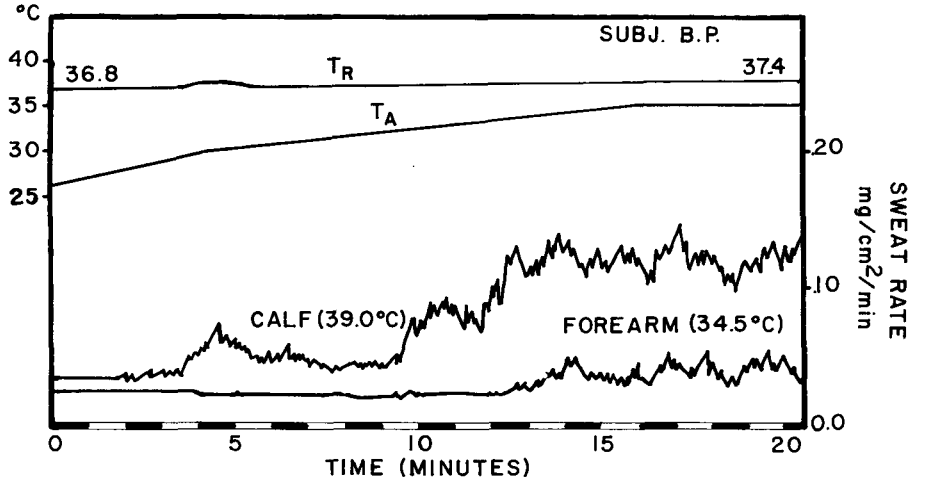

Fig. 1. The effect of different local skin temperatures on the latency and central threshold temperature in a resting subject.

the forearm at this temperature. At zero time the skin area under the sweating capsule on the calf was locally heated and maintained at 39°C. At the same time the room temperature was raised to approximately 35°C. It can be seen that the locally heated area on the calf not only showed a greater magnitude of sweating in response to the increased room temperature, but also showed a significantly shorter latency for the onset of sweating. The locally heated area showed a latency for the onset of cyclic sweating of only 2 min while the non-heated control area did not show any significant cyclic sweating activity until 10-12 min following the initial rise in room temperature. The onset of sweating in the non-heated area occurred at a significantly higher rectal temperature of 37.4°C. Similar results have been obtained in 10 different experiments on three different subjects. Since the central thermal drives to the two skin areas at any specific time during the experiment are the same, the difference in the onset of sweating in the heated area must be due to a local skin temperature effect.

The effects of local skin temperature upon the onset of sweating are illustrated in a different way in Fig. 2. In these experiments the subject was allowed to equilibrate at a room temperature of 26.0°C. Sweat capsules were place on three different skin areas and each was maintained at a different local skin temperature. One area on the forearm was locally heated and maintained at 36.1°C. Another area on the calf was locally heated to 34.5°C and a final area on the forearm was maintained at 33.1°C. At zero time the subject started working for 7 min on a bicycle ergometer. It was consistantly observed that under these ex-

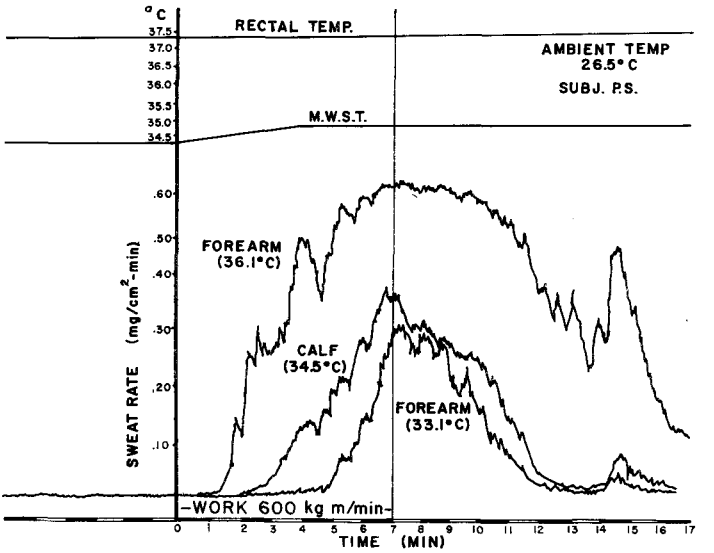

Fig. 2. The effects of three different local skin temperatures on the onset of cyclic sweating during moderate exercise.

perimental conditions the skin area with the higher local skin temperature showed a significantly greater magnitude of sweating. The areas with progressively lower local skin temperatures responded with progressively lower sweating rates. The significant point here, however, is that the latency for the onset of sweating in the 36.1°C heated area was only 1 min and was associated with a mean skin temperature of 34.5°C.

The latency for the onset in the cooler areas was progressively longer being approximately 3 min for the calf (34.5°C) and 4 min for the forearm area maintained at only 33.1°C. The onset of sweating in these areas was associated with significantly higher mean skin temperatures. It is obvious from these observations that sweat glands receiving identical central inputs can show different latencies and threshold temperatures for the onset of sweating depending upon the local temperature conditions.

CHOLINERGIC AGENTS AND THE ONSET OF SWEATING. A series of 6 experiments was done to assess the effects of increased availability of neuroglandular transmitter upon the onset of sweating. In these experiments the subject was placed in a cool room maintained at 20°C for 30 minutes. After 25 min in the cold room the subjects right forearm received an intradermal injection of pilocarpine or acetylcholine. The subject then entered a warm room maintained at 40°C and the onset of cyclic sweating on both forearms was monitored by resistance hygrometry. Figure 3 illustrates the results obtained in one of these experiments. It can be noted that in this experiment the pilocarpine treated right forearm showed a latency for the onset of cyclic sweating of 12 min, while the untreated control area on the left forearm showed a much longer latency of approximately 22 min. Furthermore, it was consistantly observed that the control and peripheral body temperatures at the onset of cyclic sweating were significantly higher in the control areas as compared with the cholinergic treated areas. It would seem, therefore, that as is the case with local heating, the local treatment of the skin with cholinergic drugs not only enhances the magnitude of cyclic sweating but also significantly effects its detectable onset and the threshold body temperatures associated with the onset.

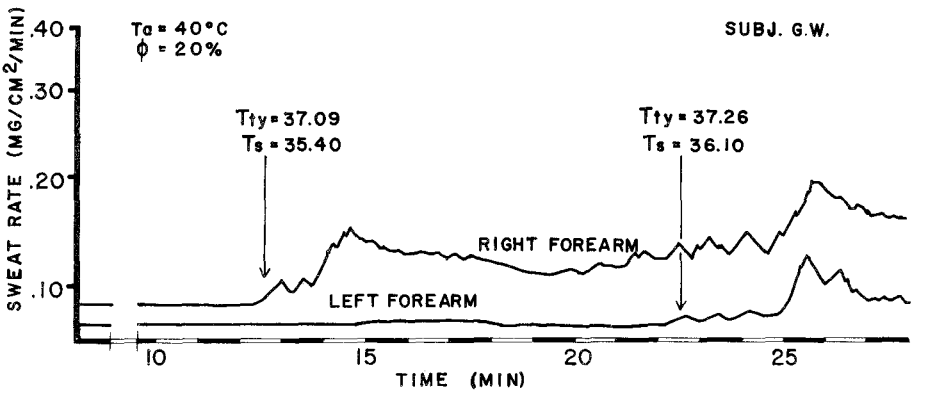

Fig. 3. The effect of an intradermal injection of pilocarpine on the onset of cyclic sweating. The arrow on the left marks the onset and corresponding temperatures for the onset of sweating in the pilocarpine treated area on the right forearm. The arrow on the right denotes the same for the control area on the left forearm.

POTENTIATION OF SUBLIMINAL IMPULSES. The responses illustrated in Figs. 1, 2 and 3 suggest the possibility that under certain conditions the eccrine sweat glands may be receiving subliminal central impulses which fail to produce active sweating. Furthermore, that these subliminal central inputs may be potentiated by local conditions at the level of the sweat gland and thus markedly effect the onset of cyclic sweating.

In order to investigate this possibility the experiment illustrated in Fig. 4 was performed. The subject was placed in a cool room at 29.6°C and 42% relative humidity. Sweating capsules were placed on both forearms and as illustrated by

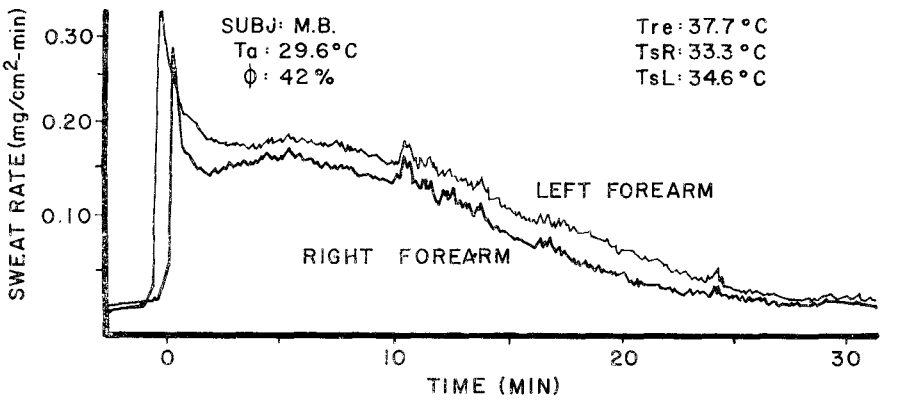

Fig. 4. Potentiation of central subliminal impulses following pilocarpine injection into both left and right forearm skin sites. Note synchrony of sweat expulsions.

the initial segment of the record no cyclic sweating was recorded. At zero time both the right and left forearms received an intradermal injection of pilocarpine. The increased availability of the cholinergic agent resulted in the production of

small sweat expulsions on both treated areas of the forearms. The sweat expulsions on the two areas were synchronous suggesting potentiation of subliminal inputs from the central thermoregulatory centers. Similar experiments have been carried out at ambient temperatures ranging from 20° to 30°C. In general, as the ambient temperature increases, the frequency of the synchronous impulses is greater and the amplitude of each expulsion is also greater.

Figure 5 illustrates the role of local skin hydration upon the effectiveness of subliminal impulses. In these experiments the subjects were equilibrated in a warm room until generalized sweating was established. At this time a wet gauze was taped to the left forearm and the ambient temperature was decreased to a subthreshold temperature. After 40 min the gauze was removed, the left forearm skin area was gently wiped and mounted with a hygrometry skin capsule. The drying curve on the left forearm is compared with the record obtained simultaneously from a pilocarpine treated area from the opposite right forearm. It can be noted that sweat expulsions which were synchronous with those observed in the pilocarpine treated area could be recorded from the hydrated area on the left forearm.

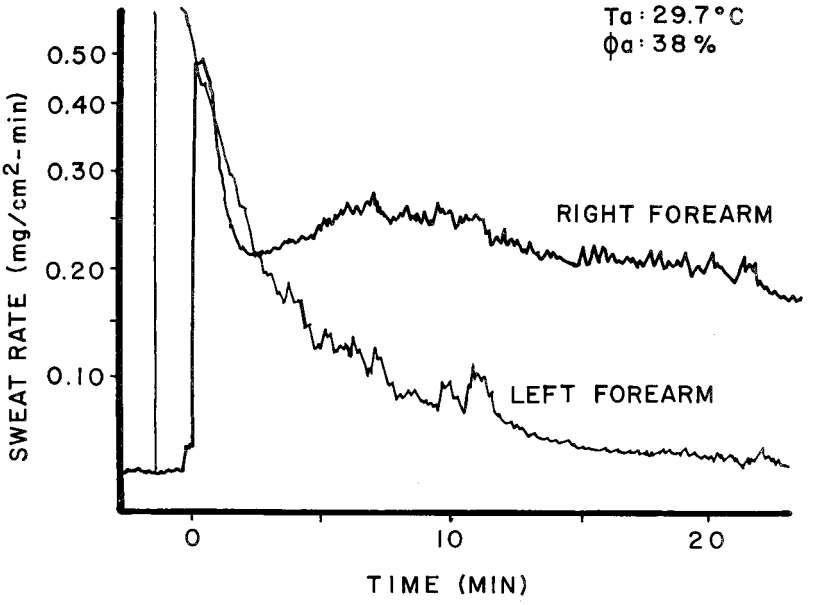

Fig. 5. Potentiation of central subliminal impulses by increased skin hydration on the left forearm. Note synchrony of sweat expulsions on the left forearm with those of a pilocarpine treated area on the right forearm.

These observations strongly suggest that some form of central sub-effective stimulation of the sweat glands is occurring at ambient temperatures between 20° and 30°C where obvious cyclic sweating is not normally observed. The effectiveness of these subliminal impulses, however, can be potentiated by local conditions such as cholinergic drug availability and the degree of local skin hydration.

SHORT TERM ACCLIMATION AND THE ONSET OF SWEATING. A series of threshold experiments was performed on three subjects to investigate the

relationship between the onset of sweating on the forearm with respect to time and threshold temperatures before and after short term acclimation. The acclimation procedure used was a modification of the technique described by Lind and Bass (1963). In these experiments the subjects were first allowed to equilibrate for one hour in a cool room maintained at 25°C. At the end of the equilibration period the subjects were quickly transferred into a climatic chamber maintained at 38°C and 20% relative humidity.

A typical threshold experiment is illustrated in Fig. 6. This figure illustrates a continuous record of the initial sweating activity on the forearm. The activity on the right side of the figure was obtained on subject P. C before heat acclimation

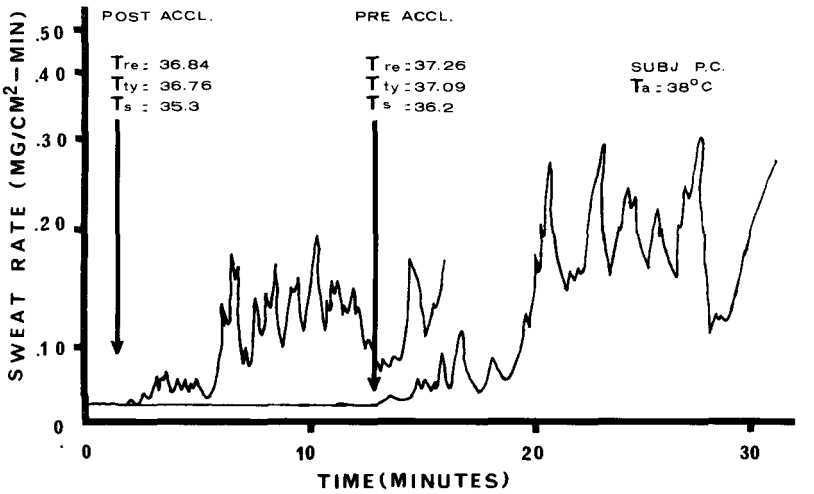

Fig. 6. Threshold temperatures and latencies for the onset of cyclic sweating in the same subject before and after heat acclimation.

while the activity on the left side illustrates the activity on the same subject after heat acclimation. It can be seen that in the pre acclimation experiment no sweating activity was recorded from the forearm during the first 13 min as indicated by the straight insensible water loss line. The body temperatures recorded at the onset of sweating in this experiment are illustrated above the arrow. The rectal temperature was 37.26°C, the tympanic was 37.09°C and the temperature of the skin as recorded on the inner thigh was 36.20°C. The record on the left side of Fig. 6 illustrates the sweating activity on the forearm of the same subject after heat acclimation. The latency for the onset of sweating after acclimation was only 3 min and was significantly shorter than in the pre acclimation experiment. The rectal temperatures at the onset of sweating was 36.84 ° while the tympanic and skin temperatures were 36.76° and 35.3°C respectively. All of these threshold temperatures were significantly lower than in the pre acclimation experiment. Similar results have been obtained in 12 other experiments and the results tend to show that the onset of sweating and the corresponding threshold temperature can be significantly effected by the degree of heat acclimation.

DISCUSSION

The role of central body temperature and the resulting hypothalamic temperature in the regulation of human eccrine sweating has been well established. The central control being mediated to the sweat glands through cholinergic sympathetic nerves (Kuno, 1956). After some years of controversy, a marked effect of several local determinants upon eccrine sweating activity has also been established. It is now quite clear that conditions at the level of the microenvironment of the glands such as local skin temperature, skin hydration, and the availability of neuroglandular transmitter substance can all markedly effect the sweating response to a constant input from the central thermoregulatory center.

In the present study we have presented evidence, Figs. 1 and 2, that local heating not only increases the magnitude of a sweating response but it can also significantly decrease the latency and threshold body temperatures for the onset of cyclic sweating as measured by resistance hygrometry during transient experimental conditions. The increased sweating response associated with local heating is not simply a result of increased glandular metabolic rate, or Q_{10} effect, but appears to be related to an increase in the amount of transmitter substance released for each neural impulse arriving at the neuroglandular junction, and an increase in the sensitivity of the sweat glands to cholinergic transmitter substances (MacIntyre et al., 1968; Ogawa, 1970). As illustrated in Fig. 3 local intradermal injection of cholinergic sudorific agents also significantly increased the sweating response, and decreased the latency and the central and peripheral threshold temperatures for the onset of cyclic sweating.

Since the central thermal drives to all areas of the skin at any specific time during our experiments are the same, the observed differences in the latency and threshold temperatures in different skin areas must be due to the alterations in the microenvironment of the sweat gland itself. Furthermore, our results indicate that during certain conditions the eccrine sweat glands may be receiving subliminal central impulses which fail to produce active sweating. As illustrated in Figs. 4 and 5 the effectiveness of the subliminal impulses may be significantly increased by cholinergic agents and increased skin hydration. It would seem that differences in the latency and threshold temperatures obtained during transient thermal conditions can be explained largely by potentiation of subliminal impulses by local glandular conditions.

Short term heat acclimation consistantly results in an increase in the secretory capacity of human eccrine sweat glands. The increased secretory capacity is related to a combination of functional changes at the level of the central thermoregulatory center (Wyndham, 1967) and at the local level of the sweat gland (Collins et al., 1966; Fox et al., 1964). These functional changes as illustrated in Fig. 6 result in a significant decrease in the latency for the onset of eccrine sweating and a decrease in the tympanic, rectal, and skin threshold temperatures for cyclic sweating. We would suggest the possibility that the decreased latency and decreased threshold temperatures for cyclic sweating following short term acclimation could be due in part to a potentiation of subliminal impulses at the level of the sweat gland due to a greater availability of neuroglandular transmitter substance.

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